Acute Necrotizing Phlegmonous Gastritis

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VERY FEW CASES of acute phlegmonous gastritis, a disease associated with a 90 per cent mortality rate, have been reported, perhaps an indication that the condition is rare. Even fewer cases have been reported in the past ten or fifteen years, possibly owing to the advent of antibiotics of various kinds.

Acute phlegmonous gastritis is a condition in which the stomach becomes so inflamed that the patient becomes severely ill, with the principal subjective symptoms referable to the epigastrium. Epigastric pain, tenderness and, sometimes, rigidity are present. Fever, nausea and vomiting usually accompany the pain. Leukocytosis occurs in most cases. So bizarre are the results of x-ray studies of the stomach that an exact diagnosis cannot be made from them. Films taken after ingestion of barium will usually show atony of the stomach and retention of the barium. Inflammation may be diffuse throughout the stomach or localized to portions of it. It does not appear to extend beyond the pylorus. In severe phlegmonous gastritis, all the stomach wall is involved and the inflammation may be so intense that the intrinsic blood supply to the organ is impaired, the wall becoming necrotic—this even though the extrinsic blood supply is normal. The exact cause for the acute inflammatory condition is not definite. Some investigators believe that it is bacterial in origin, possibly secondary to bacteremia. Hemolytic streptococcus has been implicated.2 Perhaps the inflammatory process is due to a "sensitivity" of some type.

REPORT OF A CASE

The patient was a 54-year-old white, married woman. She was admitted to the hospital with a history of three or four days of burning epigastric pain accompanied by nausea and vomiting. There was no melena. Bowel movements had been normal. Upon physical examination, tenderness in the epigastrium was noted, but no rebound tenderness or rigidity. No organs or masses were palpable. Leukocytes numbered 9,000 per cu. mm. Results of all other laboratory examinations, including a liver function test and amylase and lipase studies, were within normal limits.

Upper gastrointestinal tract x-ray studies showed some atony of the stomach and retention of barium, but no true intrinsic or extrinsic lesion was apparent. Gastric suction and supportive care were carried out, the patient improving enough to leave the hospital after nine days. Seven days later she was readmitted with symptoms similar to those of the first admission. The body temperature was

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100°F. The only positive physical finding was tenderness in the epigastrium. Leukocytes numbered 11,200 per cu. mm.—88 per cent polymorphonuclear cells. The hemoglobin was 14 gm. per 100 cc. X-ray studies of the stomach again showed atony and retention of barium. No peristalsis was recognizable in the distal half of the stomach. Two days after hospital admission the patient was operated upon. When the abdomen was opened omentum was noted to be adherent over the anterior wall of the stomach. It was freed, and the anterior wall of the stomach was observed to be necrotic, with a hole in it. The entire distal half of the stomach was brownish-black. The necrotic area extended to the pylorus, but not beyond. The mucous membrane was necrotic and had sloughed away. The proximal portion was thick-walled and slightly dusky but not necrotic. The extrinsic blood supply to the stomach was unimpaired; good pulsation could be felt in the gastric arteries. The distal two-thirds of the stomach was resected. The cut edge of the proximal portion was thick walled and edematous and bleeding was minimal. Only a slow ooze of dark blood occurred at this edge. An isoperistaltic, anticolic anastomosis was performed between the stomach and proximal jejunum. Catgut sutures were used throughout. A culture of the interior of the stomach showed Escherichia coli, aerobacter aerogenes and streptococcus. Postoperatively, the patient received chloramphenicol, 0.5 gm., every 12 hours for seven days. In addition to the usual care after gastrectomy, she received hydrocortisone sodium succinate (Solu-cortef®) intramuscularly for five days and then triamcinolone (Aristocort®) orally. The postoperative course was uneventful except for leukocytosis: 31,450 per cu. mm. on the sixth day after operation, then for the next three days 20,350, 36,000 and 13,000. The patient was discharged from the hospital on the eleventh postoperative day. Thereafter she was asymptomatic. Temperature and blood cell counts were within normal limits. She received no medication other than vitamins. In the first six weeks postoperatively she gained ten pounds in weight.

Pathologist's Report

"In all areas of the stomach examined there was extensive ulceration of the mucosa, with dense acute and chronic inflammatory cell infiltration and necrosis throughout all layers of the wall. Within the muscular coat of the stomach were areas of complete thrombotic occlusion of large arteries, associated with early organization and with acute inflammatory cell infiltration of their walls. Also present in smaller arteries were areas of older thrombosis, with complete organization and recanalization. These changes suggested that the arterial lesions antedated the inflammatory process and in most probability were the underlying cause of the ischemic necrosis. The arterial lesions did not fit

a set pattern of the more easily recognized arteritides. There was no evidence of specific inflammation or neoplasia."

SUMMARY

A patient with extensive necrotizing phlegmonous gastritis in which the intrinsic blood supply of the stomach was impaired while the extrinsic supply was normal, is presented. The major portion of the stomach was removed and the patient had no further symptoms after recovering from the operation.

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REFERENCES

- Majnarich, G., and Pawlowski, J.: Phlegmonous gastritis, Amer. J. Gastroenterol., 34:183, 1960.
 Mortland, H., and Eisenberg, D. S.: Archives Pathology, 8:744, 1929.

